Amendments to the Claims:

Listing of the Claims

This listing of claims will replace all prior versions, and listings, of claims in the application:

1-7 (Canceled)

8. (Previously presented) A method of regulating bone strength and mineralization by using a ligand that acts on a bone density regulating transmembrane receptor encoded by an isolated DNA molecule having a nucleotide sequence encoding an amino acid sequence as set forth in SEQ ID NO: 2 wherein the method comprises a first step of providing a ligand of the transmembrane receptor and a second step of administering the provided ligand in a medically acceptable form, wherein the ligand is selected from the group consisting of a human Wnt protein, a 36 kDa frizzled related protein, and a dickkopf (dkk) protein.

9. (Previously presented) A method as described in claim 8, wherein the ligand is a human Wnt protein.

10-29 (Canceled)

30. (currently amended) A method of treating osteoporosis in a human patient, comprising: (a) providing a composition that comprises a bone strength and mineralization regulator (BSMR) effector that interacts with BSMR, selected from the group consisting of WNT1, WNT2, WNT2B/13, WNT3, WNT3A, WNT4, WNT5A, WNT5B, WNT6, WNT7A, WNT7B, WNT8A, WNT8B, WNT10A, WNT10B, WNT11, WNT14, WNT15, WNT16, the 36 kDa cysteine rich frizzled related protein Frzb--1, a cysteine rich protein from the CCN family7, Mus musculus FK506 binding protein 8, Mus musculus nuclear protein 95 (Np95); GLI-Kruppel family member GLI3, Mus musculus RAN binding protein 9, Mus musculus ISL1 transcription factor, Human signal-transducing guanine nucleotide-binding regulatory (G) protein beta subunit, Mus musculus, casein kinase II, Homo sapiens zinc finger protein 198, Mus musculus, eukaryotic translation elongation factor 2, M. musculus P311, Homo sapiens E2a--Pb.times.1-associated

protein, Homo sapiens NADH dehydrogenase (ubiquinone) Fe--S protein 8, Human Smad anchor for receptor activation (SARA), Homo sapiens AMSH, and ATP6B2; and (b) administering a quantity of the composition from step (a) to the patient that is sufficient to increase alkaline phosphatase activity of bone forming cells.

31. Canceled

32. (currently amended) The method of claim ~~31~~ 30, wherein the BSMR effector is a WNT protein.

33. (previously presented) The method of claim 30, wherein the BSMR effector is complexed with a targeting moiety that concentrates the effector at one or more bone producing regions after administration to a patient.

34. (previously presented) The method of claim 33, wherein the targeting moiety is selected from the group consisting of a tetracycline, calcein, a bisphosphonate complex, polyaspartic acid, polyglutamic acid, an aminophosphosugar, a peptide known to be associated with the mineral phase of bone, osteonectin, bone sialoprotein, osteopontin, a bone specific antibody, a binding site fragment of a bone specific antibody, and a protein having a bone mineral binding domain.

35. (currently amended) The method of claim 30, further comprising an additional step of administering a ~~second~~ morphogenetic protein at least 24 hours prior to step (b).

36. (currently amended) The method of claim 35, wherein the ~~second~~ morphogenetic protein is selected from the group consisting of bone morphogenetic protein, bone morphogenetic protein 2, bone morphogenetic protein 3, hedgehog protein, endothelial growth factor, and TGF--beta 26.

37. (previously presented) The method of claim 9, wherein the WNT protein is WNT1, WNT3A or a combination thereof

38. (previously presented)The method of claim 32, wherein the WNT protein is WNT1, WNT3A, or a combination thereof